PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated meeting, February 27, 1893.

President, Dr. CHARLES K. MILLS, in the chair.

Dr. CHARLES L. DANA, of New York, read a paper entitled:

A CLINICAL AND PATHOLOGICAL STUDY OF SHAKING PALSY, WITH A REPORT OF TWO AUTOPSIES.

In the clinical part of his paper, Dr. Dana gave the results of examinations of the blood, using Ehrlich's stains; these showed the presence of a progressive chlorosis, but not an anæmia; also some changes in the myelocytes. A number of sphygmographic tracings of the pulse were shown, and the conclusion reached, that there is in paralysis agitans, a general vaso-motor paralysis affecting the skin, viscera, nerves, and lower nerve-centres, but not the brain.

In the pathological part the author first went over the literature of the pathological anatomy of paralysis agitans giving a summary of the findings in all authentic cases. The number of available cases was found to be only fourteen. The findings in late years were much more uniform and definite. The author then reported his own two cases, and gave the results of his microscopical examinations. These, in brief, revealed the presence of great vascular dilatation, connective tissue, proliferation, and nerve-cell degeneration. The lesions were most marked in the central gray of the cord, and the vagus, and glossopharyngeal nuclei. The brain cortex, internal capsule, and basal ganglia were normal.

Dr. Dana believed the vascular and connective tissue changes to be primary, the nerve-cell changes to be secondary. He thought the process resembled a low grade of chronic inflammation. At any rate, he considered the primary cause of paralysis agitans to be a toxine of autochthonous or microbic origin. Shaking palsy was a toxine disease. The primary seat of the trouble was in the spinal cord, more particularly in the central and anterior gray matter, supplied by the central arteries.

Dr. Dana thought that the time was past when paralysis-agitans could be called a functional disease without a morbid anatomy. The most recent and thorough post-mortem studies showed results in conformity with those reported by the writer. The practical conclusions in regard to therapeutics, were that remedies for the disease in in its earlier stages should and probably would be found. Such remedies would be of the character of anti-toxine or diathetic drugs modifying metabolism and promoting excretion. Some results of the author's therapeutical experience were given.

DISCUSSION.

Dr. FRANCIS X. DERCUM.—I infer from what Dr. Dana has said that he places on the changes which he finds in paralysis agitans rather the significance of terminal than the significance of causal changes. One of the points about his paper that strikes me as subversive of our former ideas is that he describes the disease as of spinal origin, and that we have a tremor of spinal and not of cortical origin. Tremor has always been looked upon as indicative of cortical and not of spinal disease.

We are still absolutely in the dark as regards the primary cause of this so-called functional trouble. Dr. Dana believes the cause to be a toxine, but this is evidently speculative only. It is to my mind fitting that in a disease in which we have motor symptoms, that we should have terminal changes in the motor tract. We have, as Osler pointed out in a paper read before this Society, terminal changes in the motor tract in other diseases such as the hopeless chorea of adults. It may be that there is some poison circulating in the blood, which leads to these peculiar changes, but there is as yet no evidence of it.

Dr. JAMES HENDRIE LLOYD.—It seems to me that the vital suggestion in Dr. Dana's paper is that of a microbic or humoral pathology of this disease. We recall that first hydrophobia, then tetanus and more recently chorea have come within the domain of the modern microbean pathology. It remained for Dr. Dana to come to Philadelphia and give us this original suggestion of some vito-chemical change in the blood; probably of microbic origin, being the cause of shaking palsy.

I cannot help thinking that some of the changes which Dr. Dana has demonstrated are rather commonplace, and possibly are identical with what we might find in the cords of many persons of the age of his patient. They certainly are not suggestive of any very distinctive disease process. Slight thickening of the blood vessels, increase in the neuroglia, and increase of the epithelium of the central canal, are rather indefinite conditions on which to establish a distinct disease process. They are terminal changes which might be attributed to a variety of irritants in the blood. The one vital suggestion is as to the possibility of this being of vito-chemical or microbic origin. The proof of the correctness of this hypothesis must be sought in systematic experimentation in the biological or bacteriological laboratory. At present it is simply an ingenious hypothesis resting upon not very broad anatomical grounds.

Dr. JAMES TYSON.—In considering the toxic origin of paralysis agitans, one is met with the thought that in diseases of acknowledged toxic origin, we have ordinarily a shorter duration than in this class of cases, with the exception of lead poisoning; while it might be expected, also, in such a disease as paralysis agitans, that the large number of cases observed would have permitted one to have established some such causal relation, if it were present.

Dr G. BETTON MASSEY.—After hearing this interesting account of the pathological changes, I feel that, personally, I shall approach the treatment of paralysis agitans with more hope of effecting results. I was one of those who believed that there was no pathology recorded in literature, and, from what I have observed, the treatment of others seems to be based on the same view, being directed toward a peripheral effect, whether internal or external remedies are used, or electricity employed. Attempts are often made to lessen the irritability of the centres by the use of Franklinic sparks, and I have seen pain apparently lessened by that form of treatment, but no arrest of the progress of the disease.

This new suggestion in regard to the microbic origin of the disease, while very interesting, does not help us a great deal, since we are already face to face with the fact that but few microbic diseases are capable of being treated directly, in spite of a positive knowledge of their microbic origin.

It appears to me that we must direct our treatment towards the removal of the result of microbic action by stimulating nutritive changes. If further attempts are to be made in the electrical treatment of this disease, we should direct our work to the passage of strong currents through the spine. I am continuously increasing the strength of currents used to the nerve centres of the spine, and I feel sure that therein we have a large field of usefulness. We have also, in these interpolar electrical applications, a means of carrying drugs into the body, by the recently revived process of cataphoresis, by which a more profound impression may be made on internal morbid processes.

Dr. J. MADISON TAYLOR.—I would ask Dr. Dana, if, in his study of this disease, there have been revealed to him any points bearing upon the early recognition of this affection and its differentiation from other forms of tremor. If there is any hope of doing good in such a disease as this, it will be in the early stages before degenerative changes have taken place.

Dr. A. A. ESHNER.—I could wish that Dr. Dana had used the term intoxicant rather than toxine in speaking of the etiology of this affection. The development of a toxine seems to commit one to the view of a microbic origin. Probably a better word still, in the present state of knowledge, would be "irritant." It seems to me that the disease belongs to the degenerative period of life, in which changes analogous to those described occur in many organs. One cannot, therefore, be surprised to find in the cord changes similar to those which we find in spleen, kidney, liver, and blood-vessels. I should have been glad if the lesions found had conformed with those of a diffuse sclerosis, affecting not only the cord but the cerebrum as well, and not necessarily confined to any group of cells or system of fibres. In conformity with this line of thought, a vascular irritant would have suggested itself in an etiologic connection. Still another point. Dr. Dana stated that paralysis agitans presents no cerebral symptoms, but it seems to me that there are occasionally cerebral symptoms of which impairment of memory is, perhaps, the most prominent. The delayed cerebral psychic tremor, sometimes noticeable, may, perhaps, be explained by an interference with the transmission of psychic impulses, as a result of changes in the conducting fibres analogous to those that are conceived to be responsible for the muscular tremor by interfering with the transmission of motor impulses. It seems to me that by the assumption of an intoxicant or irritant, dependent upon defective elimination by the emunctories, we need not extend the range of speculation to such a limit as to have to include a possible microbic origin.

The PRESIDENT.—It seems to me that in some respects the discussion is hardly just to the paper. Some of the suggestions are new. We cannot say that only terminal or the ordinary degenerative changes have been described. The histological changes of degenerative disease will almost necessarily be the same or similar, however they may be described. Dr. Dana has suggested to us something definite. The extent and the location of the most intense changes which he found are different from those found in other degenerative diseases. The suggestion as to place of initiation of the changes; the question of the degeneration of the lateral columns of the cord; the fact that he has shown that in this one case at least no definite changes were found either in the cortex or in the cerebral tracts, or anywhere above the pons, and also that he has pointed out that the changes were especially marked in the nuclei of certain of the cranial nerves, are of considerable importance, and some of them have a decided bearing upon the peculiarities of symptomatology.

Dr. C. L. DANA.—When gentlemen now-a-days speak scornfully of microbe and microbic products, I am reminded of the Rev. Mr. Jasper, and his assertion that "the sun do move." And I am content to allow those who enjoy thinking that microbes are still theoretical things to join the ranks with the Rev. Mr. Jasper. I believe thoroughly in their pathogenic importance myself, and there is nothing of late years that has advanced us so much as bacteriology, or that gives us so much hope for the future of therapeutics. At the same time, I did not say that paralysis agitans was a microbic disease. I said that it probably was a toxic affection, and that the toxine was either of microbic or of humoral origin; and by humoral origin, I mean the same kind of origin as is supposed to produce the changes in rheumatism and gout, which we take to be the result of some defect in tissue metabolism or glandular activity.

These changes have been spoken of as terminal changes. All anatomical changes are terminal. The changes which you find in the lungs in pneumonia are terminal. They do not cause pneumonia. There is something that causes the anatomical changes. No one thinks that pneumonia is caused by the presence of leucocytes in the air cells. I fancy also that the changes in Bright's disease are terminal, due to the constant irritation of the parenchyma of the kidney.

It has been intimated that the changes which I have described as occurring in paralysis agitans are of the same nature as those found in senility. I have considered this point in the paper, but did not dwell upon it when reading. My own experience is that the changes are not the same. I have examined the cords of old persons, and have compared them with the cords from these cases. The arteries are not thickened here as in senility, and there are peculiar changes in the cells. I think that the changes are such as would be produced by an irritant acting upon special parts of the spinal cord.

I can only say with reference to tremor that, so far as my experience goes, the tremor produced by irritation of the cortex of the brain, is a fine tremor of vibratory character, and that, when a coarser tremor has been observed, it has been due to some lesion lower down. I have had cases of myelitis where there was distinct coarse tremor like that seen in paralysis agitans.

Adjourned.